thirds of normal. There may thus be substantial impairment of renal function even in patients in whom the blood urea level is normal. Three points follow from this: (1) That the chosen dose of contrast medium should err on the generous side because one often cannot tell which patient will have some impairment of function. (2) That in the aged, in whom renal function normally deteriorates, the dose should be increased. (3) That even minor elevation of the blood urea should be communicated on the X-ray request card so that the dose can be adjusted accordingly.

By the time there is renal failure the GFR is grossly reduced. This is not, however, the only factor affecting the quality of the films in renal failure. The second point of considerable importance in these patients is that they are unable to concentrate their urine. This defect in concentration is due not only to tubular damage but also to the diuresis caused by the osmotic load associated with the high level of blood urea. This raises a point of practical importance: an attempt to improve the IVP by depriving these patients of fluids can do no good and may do harm, as they continue to lose water and may get a serious disturbance of fluid balance. In the face of the grossly reduced GFR, the tubular damage and the added osmotic diuresis of renal failure, the only way to obtain some opacification is to raise the plasma level very high by giving large doses. We ourselves at present give a dose corresponding to approximately 1 ml/lb Hypaque 45 and would expect to obtain useful information in patients with blood urea levels up to 200 mg/100 ml and often much higher.

One way of administering such a dose is by drip infusion pyelography. The most important aspect of the technique is the large dose. Apart from its use in patients with renal failure, such a dose has other uses, as for instance when it is necessary to observe a dense nephrographic effect or when it is desired to fill both ureters well. With regard to the calyces, it is our experience that in patients with normal renal function they are adequately shown with about half the dose; indeed they may be better shown due to a less dense nephrogram and less over-filling (Dure-Smith 1966). The technique of injection itself with the addition of dextrose saline has in our own experience made no difference to the quality of the IVP when compared with normal intravenous injection; it is best regarded simply as a convenient means of giving a large dose.

Finally, I would like to emphasize three points: (1) The main variables concerned in excretion of contrast medium during an IVP can conveniently be thought of under three main headings, plasma level, GFR and the factors affecting the amount of water reabsorbed. (2) It is tempting to use the IVP as an assessment of renal function, but there are so many different factors concerned in the final image of the collecting system that any such attempt must be made with great caution and can only lead to a very crude assessment. (3) IVP techniques have increasingly to be tailored for a particular patient with a particular problem. Knowledge of the variables involved makes it easier to plan what to do in a given situation. More than ever is close co-operation required between the clinician and the radiologist if the patient is to be given the most effective investigation.

REFERENCES
Cattell W R, Fry I K, Spencer A G & Purkiss P (1967) *Brit. J. Radiol.* **40**, 561
Dure-Smith P (1966) *Brit. J. Radiol.* **39**, 655
Fry I K, Cattell W R, Spencer A G & Purkiss P (1967) *Brit. J. Radiol.* **40**, 572
Purkiss P, Spencer A G, Cattell W R & Lane R D (1967) (in preparation)
Saxton H M & Strickland B (1964) Practical Procedures in Diagnostic Radiology. London; p 32

Nephrotomography Simplified

by H M Saxton MRCP FFR
(*Guys Hospital, London*)

High dose excretion urography produces a relatively dense nephrogram due to the high tubular concentration of contrast. The opacification occurs in functioning parenchyma and so may be employed to demonstrate alterations in function or anatomical disturbances in a way paralleling the use of the nephrographic phase in aortography (angionephrogram).

The Physiological Basis of the Nephrogram (Fig 1)
The nephrogram is produced by contrast along the whole length of the nephron. Except in the collecting tubules the concentration of contrast in the tubule will vary according to that in the glomerular filtrate. It therefore depends on the plasma level of contrast and is affected by the glomerular filtration rate. Thus the densest nephrogram is seen immediately after injection of a large dose of contrast in a patient with normal renal function. Renal failure, including the

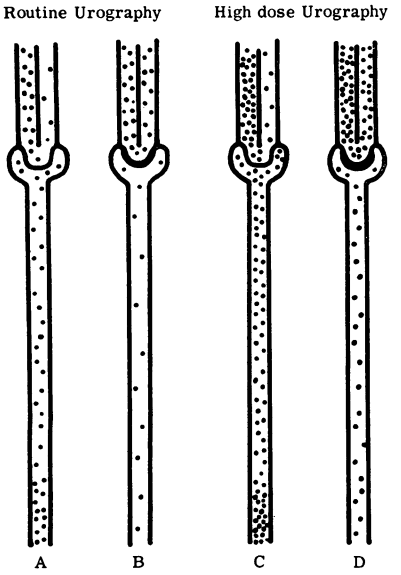

Fig 1 *Diagram to show, in terms of a nephron, how the kidney as a whole handles contrast. The area representing the tubule is the source of the nephrogram. (Routine urography, e.g. 40 ml of 45% Hypaque. High dose* – see text.) A, *normal kidney, patient dehydrated: normal filtration provides a moderate tubular concentration and some nephrographic density; further concentration in the collecting tubules.* B, *renal failure: reduced filtration leads to low tubular concentration and poor nephrogram; no distal concentration.* C, *normal kidney, patient dehydrated: normal filtration gives a high tubular concentration and good nephrogram. Some further concentration in the collecting tubules.* D, *renal failure: impaired filtration but the high blood level leads to relatively high tubular concentration of contrast and so to some nephrographic density; no distal concentration*

gradual renal impairment of age (de Wardener 1961), results in a poorer nephrogram but some opacification may be possible (Fig 2). The state of hydration of the patient has relatively little effect on the nephrogram because concentration and dilution occur in the collecting tubules. Thus it is possible to obtain a good nephrogram and a poor pyelogram. (For a further discussion of the physiology of contrast excretion *see* Cattell *et al.* 1967 and Fry *et al.* 1967.)

Table 1
Conditions in which high-dose nephrotomography may be employed

Assessment of normal renal parenchyma
Fœtal lobulation
Dromedary kidney
Ectopic kidney
Rotated kidney
Horseshoe kidney
(Retoperitoneal lesions displacing the kidney)
Parenchymal disturbance or 'non-function'
Renal failure
Renal scarring
Replacement lipomatosis
Hydronephrosis
Pyonephrosis
(Renal abcess)
(Renal tuberculosis with parenchymal destruction)
(Renal infarct)
Renal masses
Polycystic disease
Cysts single or multiple
Renal tumour

Lesions listed in parentheses were not studied in the present series

Technique and Indications

The method used is a simplified form of nephrotomography (Evans *et al.* 1955, Evans 1966), no attempt being made to achieve rapid injection or to show the phase of vascular filling. Possibly vascular opacification may contribute to the overall density but since even the angionephrogram has a substantial tubular element (Edling & Helander 1959) there is little doubt that tubular opacification is the most significant component. Conray 420 (sodium iothalamate), 100 ml in an average patient, is injected at normal speed (about two to three minutes). The renal areas are then tomographed immediately. Sufficient tomographic cuts must be employed or valuable information

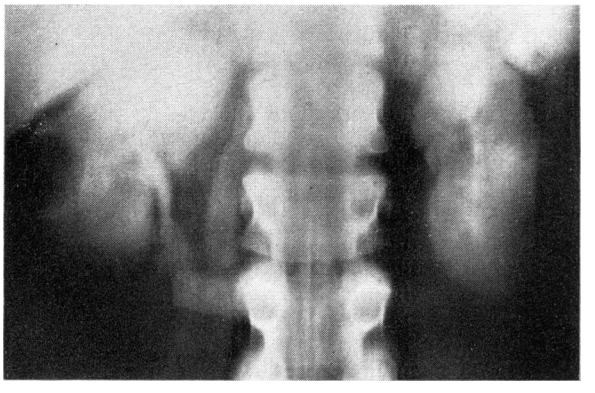

Fig 2 *Chronic glomerulonephritis. Blood urea 130 mg/100 ml. HDNT gives a good nephrogram showing a right renal cyst*

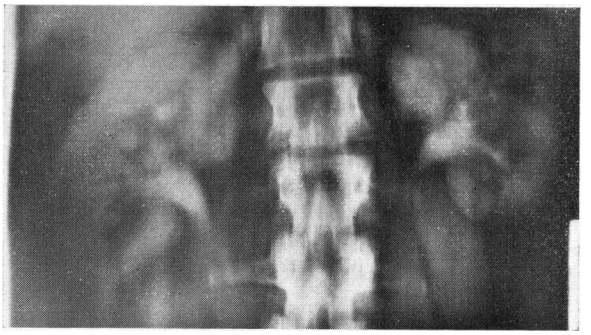

Fig 3 *Renal failure. Blood urea 80 mg/100 ml; proteinuria 10 g daily. HDNT shows renal atrophy, particularly on the left, with patchy central fat replacement*

may be lost. Preliminary tomography is not essential but is useful, particularly when tomographing in an unusual area, e.g. pelvic kidney or horseshoe kidney.

The technique is most frequently employed to give further information on a problem raised by a routine examination but is itself used as a routine on the following indications: renal failure[1], known nonfunctioning kidney, hæmaturia, renal masses or suspected polycystic disease. The full range of conditions investigated is set out in Table 1.

High-dose nephrotomography (HDNT) may be used to show that minor irregularities in renal parenchyma – most commonly fœtal lobulation – contain normally opacified tissue. Ectopic kidneys may outline poorly because of the lack of perirenal fat but HDNT shows the parenchyma well and can similarly show the functioning bridge in a horseshoe kidney.

Parenchymal Disturbances

In renal failure or scarring the state of the parenchyma can often be well demonstrated (Fig 3) if desired. The method clearly reveals the presence of renal sinus fat in some normal patients, particularly the elderly or obese (Fig 4) and also the frequent occurrence of varying degrees of renal sinus lipomatosis (Faegenburg *et al.* 1964, Crummy *et al.* 1966) in many types of renal atrophy (Fig 5). In a few cases of renal lipomatosis a characteristic calyceal deformity is produced – the so-called fibrolipomatosis (Olsson & Weiland 1963, Kreel *et al.* 1966) – and here too HDNT helps to confirm the diagnosis (Fig 6A, B). Fatty replacement may be patchy or continuous. It appears translucent compared to the opacified renal tissue and the transition from fat to parenchyma is characteristically ill defined (Faegenburg *et al.* 1964).

[1]To avoid an undue loading with sodium in renal failure the methylglucamine salt of iothalamic acid (Conray 280) should be given in equivalent dosage (150 ml)

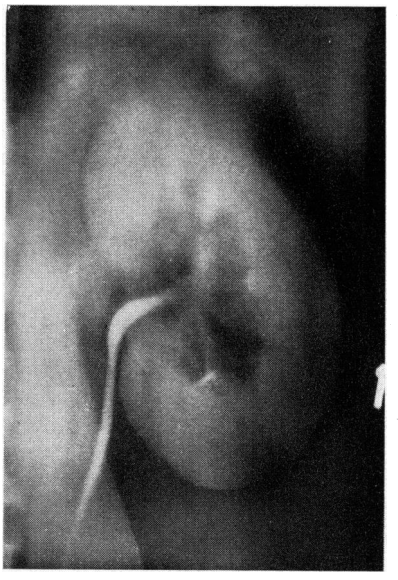

Fig 4 *Renal sinus fat in a normal obese patient*

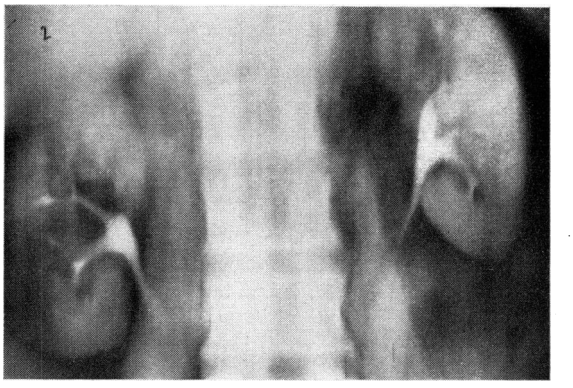

Fig 5 *Diabetic with blood urea 60 mg/100 ml, hypertension and proteinuria. HDNT demonstrates replacement lipomatosis*

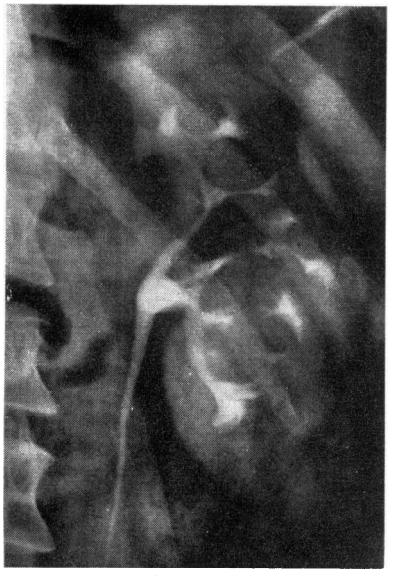

Fig 6A *Man, aged 58, with mild proteinuria and hypertension. Had severe nephrotic syndrome ten years previously. The intravenous urogram shows the deformity characteristic of 'fibrolipomatosis'*

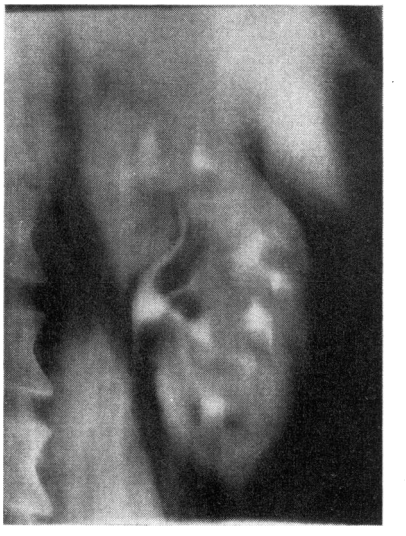

Fig 6B *HDNT confirms the central fat translucency*

Renal parenchymal opacification in obstructive disease can be of value, especially in the apparently nonfunctioning kidney. Hydronephrosis of all degrees has been demonstrated 'in reverse' (Figs 7, 8), the non-opacified urine in the distended pelvis contrasting with the rim of stretched but opacified kidney tissue. Other disorders of renal parenchyma as set out in Table 1 are likely to show as non-opacified areas within the parenchyma but no suitable cases have been encountered.

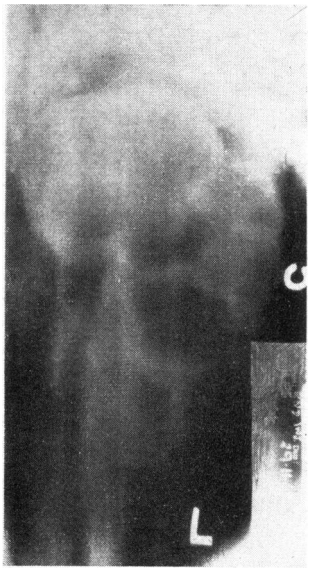

Fig 7 *Tuberculous pyonephrosis. HDNT carried out for nonfunctioning left kidney shows the distended renal parenchyma (no calcification in the operation specimen)*

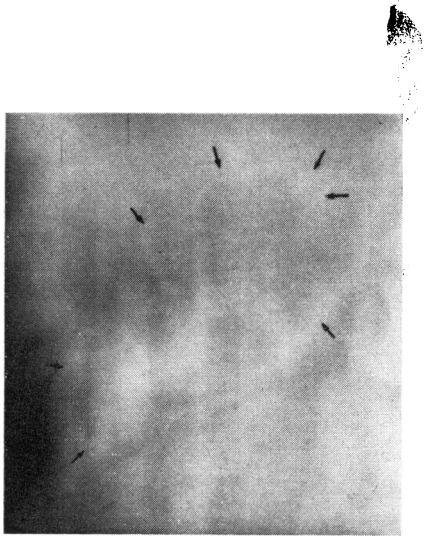

Fig 8 *Hydronephrosis following ureteric ligation at hysterectomy. Faint opacification of a thin rim of distended renal tissue; at operation this was only 1–2 mm thick*

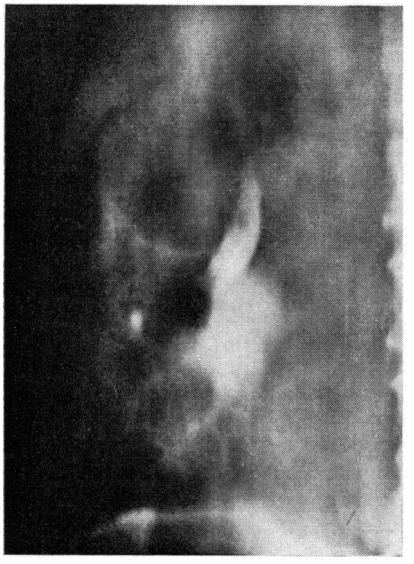

Fig 9 *Polycystic disease showing the characteristic pattern of cysts of all sizes outlined by streakily opacified parenchyma*

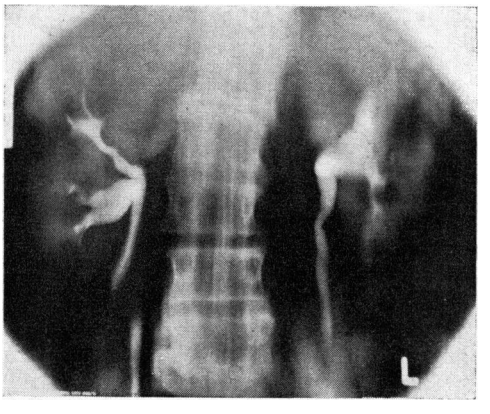

Fig 10 *Multiple renal cysts in a 71-year-old man*

Renal Masses

In polycystic disease a characteristic spongy texture is displayed (Fig 9) and the different sizes of cyst can be seen as translucencies surrounded by streakily opacified parenchyma. In multiple cysts the intervening parenchyma appears normal (Fig 10). Single cysts can be well displayed (Fig 11). When they show as sharply defined spherical translucencies, with a clear-cut margin and, if peripheral, a thin rim, the diagnosis of cyst is

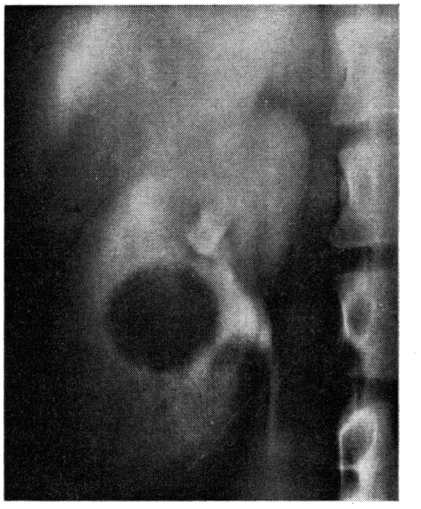

Fig 11 *Central renal cyst in a 35-year-old woman, shown by HDNT and confirmed by arteriography and cyst puncture. Note the sharply defined margin which was not so clear cut on the tomographic sections on either side of this one*

virtually certain. If Casoni and hydatid complement-fixation tests are negative it is then reasonable to proceed directly to cyst puncture. It has not, however, proved possible to achieve an overall level of accuracy comparable to the figure of 95% claimed by Evans (1966) for nephrotomography in the distinction between tumours and cysts. In the majority of cases some doubt remains and in these selective renal arteriography remains the first choice for accurate diagnosis.

REFERENCES

Cattell W R, Fry I K, Spencer A C & Purkiss P (1967) *Brit. J. Radiol.* **40**, 561

Crummy A B, Barquin O P & Wear J B (1966) *J. Urol.* **96**, 127

Edling N P G & Helander C G (1959) *Acta radiol. (Stockh.)* **51**, 17

Evans J A (1966) In: Radiographic Atlas of the Genitourinary System. Ed. C Ney & R M Friedenberg. Philadelphia & London; p 302

Evans J A, Monteith J C & Dubilier W (1955) *Radiology* **64**, 655

Faegenburg D, Bosniak M & Evans J A (1964) *Radiology* **83**, 987

Fry I K, Cattell W R, Spencer A G & Purkiss P (1967) *Brit. J. Radiol.* **40**, 572

Kreel L, Melmed R N & Bouchier I A D (1966) *Brit. J. Radiol.* **39**, 837

Olsson O & Weiland P O (1963) *Acta radiol. (Stockh.) Diag.* **1**, 1061

Wardener H E de (1961) The Kidney. London